trast to the findings of Melo and colleagues,[1] and despite the younger age of our patients, we found no significant difference in the incidence of AF between the two well-matched groups in our study.

Melo and colleagues[1] failed to come up with a convincing reason for the claimed effectiveness of the technique. Anatomically, the route taken by autonomic nerves to the heart is highly variable[2]; a lesser but significant and variable proportion of autonomic nerves reaches the heart along the pulmonary veins and the back of the heart, so the actual denervation achieved in quantitative terms is not consistent or comparable between patients unless the innervation is mapped out before denervation. The inclusion of patients with diabetes (26% in the VCD group and 27% in the control group in their study and 16% and 13%, respectively, in our own study) and patients receiving β-blockers (75% in the VCD group and 81% in the control group in their study and 69% and 72%, respectively, in our study) gives rise to additional problems. Many patients with diabetes already have partial denervation from preexisting autonomic neuropathy, and it is debatable how much additional denervation is achieved surgically with the technique. β-Blockers themselves have antiarrhythmic effects and produce a state of near-total pharmacologic sympathetic denervation. Long-term β-blockade causes β-receptor hypersensitivity, necessitating reintroduction of β-blockers after the operation. The reintroduction of β-blockers in most cases in the postoperative period results in total cardiac sympathetic denervation and partial parasympathetic denervation, which raises questions about the whole hypothesis.

One can only meaningfully assess the effectiveness of the technique through a prospective, randomized multicenter trial excluding patients with diabetes and those receiving β-blockers. Histologic quantification of the amount and type of nerve fibrils in the excised fat pads is also necessary.

Joseph Alex, MBBS, MRCS
Mazhar Ur Rehman, MBBS, FRCS (CTh)
Levent Guvendik, MD, FRCS, FETCS
Department of Cardiothoracic Surgery
Castle Hill Hospital
Hull And East Yorkshire Hospitals NHS Trust
Hull, United Kingdom

References

1. Melo J, Voigt P, Sonmez B, Ferreira M, Abecasis M, Robocho M, et al. Ventral cardiac denervation reduces the incidence of atrial fibrillation after coronary artery bypass grafting. *J Thorac Cardiovasc Surg*. 2004;127:511-5.
2. Pauza D, Skripa V, Pauzienne N, Stropus R. Morphology, distribution and variability of the epicardial neural subplexus in the human heart. *Anat Rec*. 2000;259:353-82.

doi:10.1016j.jtcvs.2004.03.044

Use of enoxaparin in cardiac surgery

To the Editor:

We congratulate Dr Medalion and coworkers[1] for the results reported in their study recently published in the *Journal* that focused on the effects of enoxaparin in patients undergoing coronary operations. The optimal administration of low-molecular-weight heparin in cardiac surgery is a controversial issue.[2,3] The use of enoxaparin in patients with prosthetic valves has also been recently described.[4,5] Because we have routinely used enoxaparin both preoperatively and postoperatively in nonemergency cardiac patients at our institution during the last 5 years, we would like to outline some aspects of our experience.

When we started using enoxaparin in 1998, we had some concerns related to the potential bleeding hazards. Although we never measured anti-factor Xa activity, we did not experience any major bleeding complications that could primarily be related to the use of enoxaparin itself, and we gradually developed a simplified protocol (Table 1), which is, however, significantly different from the one described. Doses of preoperative enoxaparin are much lower, and antiplatelet therapy is stopped on admission. On the other hand, a low dose of enoxaparin is given to all patients awaiting surgical intervention once daily (twice daily if the dose exceeds 4000 U/d). Also, patients who could potentially be stabilized with higher doses of enoxaparin and treated on an urgent basis, typically patients with coexistent left main disease and unstable angina, are instead treated as emergency cases and, as such, receive unfractionated heparin infusion until transfer to the operating room. Enoxaparin is also used after surgical intervention to minimize thromboembolic complications until a satisfactory international normalized ratio is attained in patients receiving oral anticoagulation and to prevent deep venous thrombosis.

In the study cited above,[1] although it is clear that patients classified as emergency cases receive heparin infusion, the authors do not specify when to administer subcutaneous enoxaparin versus intravenous heparin in urgent cases. The level of anticoagulation and the laboratory tests used (eg, partial thromboplastin time and activated clotting time) in patients receiving intravenous heparin are also not reported, and this might influence the measurements of anti-factor Xa activity at skin incision. Although results might be biased by a special accuracy in surgical hemostasis in patients enrolled in the study, bleeding rates were low in all subgroups despite high doses of enoxaparin (1 mg/kg twice daily),

TABLE 1. Use of enoxaparin before and after cardiac operations

Variable	Dose
Preoperative course	
Standard minimal dose	2000 U
Minimal dose in patients with atrial fibrillation or oral anticoagulation	4000 U
Maximal dose	2 × 4000 U
Conditions that modify preoperative doses	
Intravenous nitrates or left main stenosis >50%	2 × dose
Endocarditis or cardiac neoplasm	2 × dose
Body mass index >30	2 × dose
Body mass index <25	1/2 dose
Ticlopidine or clopidogrel withdrawal	1/2 dose
Hemoglobin <12 g/dL	1/2 dose
Postoperative course	
Standard dose	2000 U until mobilization
Oral anticoagulation or atrial fibrillation	4000 U until INR >2
Body mass index >30	4000 U until mobilization

One hundred units of enoxaparin = 1 mg.
INR, International normalized ratio.

administration of enoxaparin late until surgical intervention (8.7 ± 0.75 hours preoperatively), and nonwithdrawal of aspirin.

In conclusion, we agree with the authors that the use of enoxaparin before routine cardiac operations appears safe as far as major bleeding complications are concerned. Probably this consideration also applies to the early postoperative period; in this respect administration of high-dose enoxaparin (1 mg/kg twice daily) has recently been reported after mechanical valve implantation as a bridge to satisfactory oral anticoagulation.[5] Finally, higher doses might be justified in some subsets of critically ill patients undergoing coronary operations, although a clear cutoff indication between subcutanous enoxaparin versus heparin infusion remains to be elucidated. In our opinion caution is advised because the benefits of high-dose enoxaparin might be outweighed by the risks related to a less aggressive attitude toward unstable patients awaiting coronary bypass surgery.

Marco Pocar, MD, PhD
Alessio Assaghi, MD
Francesco Donatelli, MD
Cattedra di Cardiochirurgia
Università degli Studi di Milano
Policlinico MultiMedica
Milan, Italy

References

1. Medalion B, Frenkel G, Patachenko P, Hauptman E, Sasson L, Schachner A. Preoperative use of enoxaparin is not a risk factor for postoperative bleeding after coronary artery bypass surgery. *J Thorac Cardiovasc Surg*. 2003;126:1875-9.
2. Kincaid EH, Monroe ML, Saliba DL, Kon ND, Byerly WG, Reichert MG. Effects of preoperative enoxaparin versus unfractionated heparin on bleeding indices in patients undergoing coronary artery bypass grafting. *Ann Thorac Surg*. 2003;76:124-8.
3. Jones HU, Muhlestein JB, Jones KW, Bair TL, Lavasani F, Sohrevardi M, et al. Preoperative use of enoxaparin compared with unfractionated heparin increases the incidence of re-exploration for postoperative bleeding after open-heart surgery in patients who present with an acute coronary syndrome: clinical investigation and reports. *Circulation*. 2002;106(suppl I):I19-22.
4. Ferreira I, Dos L, Tornos P, Nicolau I, Permanyer-Miralda G, Soler-Soler J. Experience with enoxaparin in patients with mechanical heart valves who must withhold acenocumarol. *Heart*. 2003;89:527-30.
5. Fanikos J, Tsilimingras K, Kucher N, Rosen AB, Hieblinger MD, Goldhaber SZ. Comparison of efficacy, safety, and cost of low-molecular-weight heparin with continuous-infusion unfractionated heparin for initiation of anticoagulation after mechanical prosthetic valve implantation. *Am J Cardiol*. 2004;93:247-50.

doi:10.1016/j.jtcvs.2004.02.043

Reconstruction of double-outlet right ventricular outflow tract comprising a pulmonary artery flap in a child with an anomalous coronary artery

To the Editor:

van Son[1] provided a useful operative solution for repairing tetralogy of Fallot of anomalous origin in the left anterior descending coronary artery and small pulmonary annulus. The technique, using a pulmonary artery flap as the posterior wall of the constructed pathway, has the potential advantage of preserving growth and avoiding coronary damage for this subset. However, no interim or long-term results have been reported.

A 3-year-old boy who had been cyanotic since birth because of a double-outlet right ventricle with a subaortic ventricular defect and infundibular-valvular pulmonary stenosis (z value[2] of -3.6) underwent a definitive operation according to the instructions of the proposed technique at our institute. The right coronary artery originated from the aorta, transversing the right ventricular outflow tract (RVOT) very near the main pulmonary trunk because of l-transpositioned great arteries. Seven years postoperatively, the pressure gradient across the RVOT had increased to 80 mm Hg from 20 mm Hg, even though the native pulmonary valve annulus and a patchy reconstructed tract orifice had grown by 11 mm ($z = -2.8$) and 15.5 mm ($z = +1.2$), respectively (Figure 1). Meanwhile, the right coronary artery beneath the constructed pathway showed no distortion.

The growth of the RVOT without harmful influence on the coronary artery was proved in this case, and the problem involved in leaving the native outflow tract open was elucidated. The anatomic stenosis advanced concomitant with development of the native pathway. The bloodstreams from both pathways probably created turbulence at the merging point, making the outflow tract bulge centrifugally. The dilated native posterior wall rose up and protruded into the pathway as a transverse fold. Although we still support the early definitive repair of Fallot-type cyanotic disease, we conclude that com-

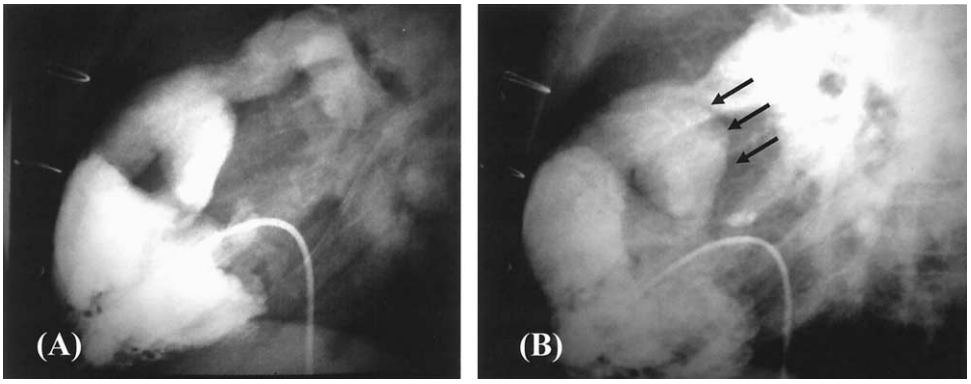

Figure 1. A, Angiogram 4 years postoperatively: the right ventricular angiogram demonstrates the development of reconstructed and native pathways. B, Angiogram 7 years postoperatively: the native outflow tract bulged centrifugally, and the posterior wall protruded into the pulmonary outflow tract as a transverse fold *(black arrows)* with a pressure gradient of 80 mm Hg.